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Comparison of two ventilatory strategies in elderly patients undergoing major abdominal surgery

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Background. 'Open lung' ventilation is commonly used in patients with acute lung injury and has been shown to improve intraoperative oxygenation in obese patients undergoing laparoscopic surgery. The feasibility of an 'open lung' ventilatory strategy in elderly patients under general anaesthesia has not previously been assessed.

Methods. 'Open lung' ventilation (recruitment manoeuvres, tidal volume 6 ml kg^{-1} predicted body weight, and 12 cm H_2O PEEP) (RM group) was compared with conventional ventilation (no recruitment manoeuvres, tidal volume 10 ml kg^{-1} predicted body weight, and zero end-expiratory pressure) in elderly patients (>65 yr) undergoing major open abdominal surgery with regard to oxygenation, respiratory system mechanics, and haemodynamic stability. We also monitored the serum levels of the interleukins (IL)-6 and IL-8 before and after surgery to determine whether the systemic inflammatory response to surgery depends on the ventilatory strategy used.

Results. Twenty patients were included in each group. The RM group tolerated open lung ventilation without significant haemodynamic instability. Intraoperative Pa_{O_2} improved in the RM group ($P<0.01$) and deteriorated in controls ($P=0.01$), but postoperative Pa_{O_2} was similar in both groups. The RM group had improved breathing mechanics as evidenced by increased dynamic compliance (36%) and decreased airway resistance (21%). Both IL-6 and IL-8 significantly increased after surgery, but the magnitude of increase did not differ between the groups.

Conclusions. A lung recruitment strategy in elderly patients is well tolerated and improves intraoperative oxygenation and lung mechanics during laparotomy.

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Atelectasis develops within minutes after the induction of general anaesthesia,[1 2] and is a significant source of intraoperative gas exchange abnormalities.[1] These areas of atelectasis can be ameliorated in part by a lung recruitment manoeuvre (i.e. sustained, high airway pressures) followed by a substantial level of PEEP,[3] which has been demonstrated to improve intraoperative oxygenation in morbidly obese patients.[4 5] This concept is similar to the 'open lung' ventilatory strategy applied to patients with acute respiratory distress syndrome.[6] This strategy aims to reduce the potential for ventilator-induced injury by minimizing stress applied to the lung parenchyma by repeated opening and closing of atelectatic lung areas which contribute to tissue disruption.[7] Open lung ventilation uses lower tidal volumes, which may further minimize parenchymal stress.[8] The potential benefits of this approach have been suggested both in terms of improving gas exchange and reducing microvascular endothelial disruption.[7 9] This

ventilation strategy may have clinical benefit in patients with acute respiratory distress syndrome. For example, one study found that it improved 28 day survival, increased weaning rates from mechanical ventilation, and decreased the frequency of barotrauma.[6]

Advanced age is a risk factor for the development of pulmonary complications, and age-related changes in pulmonary function[10] may increase the potential for intraoperative lung injury. In the awake patient, partial pressure of arterial oxygen (Pa_{O_2}) decreases with increasing age.[1] Almost all elderly mechanically ventilated patients developed atelectasis and shunt during anaesthesia.[1]

The potential utility of an 'open lung' ventilatory strategy to improve intraoperative oxygenation and to reduce lung parenchymal injury has not been studied in the elderly undergoing general anaesthesia. In this exploratory study, we test the hypothesis that an 'open lung' ventilatory strategy[11] improves oxygenation and mechanics of breathing in elderly patients undergoing open abdominal surgery. We compared an 'open lung' strategy [recruitment manoeuvres, tidal volume (V_T) 6 ml kg^{-1} predicted body weight, and 12 cm H_2O PEEP] with a group who received conventional ventilation [no recruitment manoeuvres, V_T 10 ml kg^{-1} predicted body weight, and zero end-expiratory pressure (ZEEP)]. As a surrogate measure of lung parenchymal injury, we also monitored the serum concentrations of the cytokines interleukin (IL)-6 and IL-8 before and after surgery to determine whether the systemic inflammatory response to surgery depends upon the ventilatory strategy used.

Methods

Study population

The Mayo Clinic Institutional Review Board approved this investigation, and each subject gave written informed consent. Patients aged >65 yr undergoing major open abdominal surgery at Saint Mary's Hospital, Rochester, MN, USA, were eligible for enrolment. Exclusion criteria included significant pulmonary disease with abnormalities in spirometry consistent with either obstructive or restrictive pulmonary disease, active asthma (requiring chronic bronchodilator therapy), previous lung surgery, home oxygen therapy, significant cardiac dysfunction (left ventricular ejection fraction <40%), or BMI >35 kg m^{-2}.

Anaesthetic management

A standardized anaesthetic technique was used in both groups. Anaesthesia was induced with propofol (1–2 mg kg^{-1}), fentanyl (1–2 μg kg^{-1}), and succinylcholine (2 mg kg^{-1}) or vecuronium (0.1 mg kg^{-1}) and maintained with desflurane, vecuronium, and oxymorphone. The concentration of desflurane was titrated at the discretion of the attending anaesthetist. In addition to the standard American Society of Anesthesiologists monitors, arterial pressure was measured *via* a radial artery catheter in all patients. Hypotension (mean arterial pressure ≤60 mm Hg) was treated with either ephedrine 5 mg i.v. or phenylephrine 100 μg i.v. In all patients, fluid management was left to the discretion of the attending anaesthetist. Normothermia was maintained with a forced-air warmer. None of the patients received dexamethasone (for PONV prophylaxis) or ketorolac before the second blood sample for determination of cytokines was obtained.

Ventilatory management

Patients were randomized to one of the two ventilatory management strategies using a randomization schedule provided by the Division of Biostatistics. In all patients, mechanical ventilation (Datex-Ohmeda Aestiva/5 Smart Ventilator, Madison, WI, USA) was initiated immediately after tracheal intubation using volume-control mode. In both groups, inspiratory-to-expiratory time ratio was 1:2 and inspired oxygen fraction ($F_{I_{O_2}}$) was 0.5 (balance nitrogen). In the control group, ventilator settings included a rate of 8 bpm, V_T of 10 ml kg^{-1} ideal body weight, and a PEEP set at 0 cm H_2O (ZEEP) (actual PEEP was ~2.5 cm H_2O due to the intrinsic PEEP of the mechanical ventilator). In the recruitment manoeuvre (RM) group, the ventilation settings after tracheal intubation were a rate of 8 bpm, V_T of 6 ml kg^{-1} ideal body weight, and a PEEP of 4 cm H_2O; this PEEP was maintained until the first recruitment manoeuvre. In this group, lung recruitment was achieved by sequential increases in PEEP in three steps from 4 to 10 cm H_2O (for 3 breaths), 15 cm H_2O (for 3 breaths), and 20 cm H_2O PEEP (for 10 breaths). After recruitment, the level of PEEP was maintained at 12 cm H_2O throughout the entire operation. Lung recruitment was repeated at 30 and 60 min after the first recruitment and hourly thereafter. This regimen was based on our previous work showing efficacy in improving gas exchange in morbidly obese patients.[4 5] If the end-tidal CO_2 increased in either study group above 6.7 kPa (50 mm Hg), the rate was increased while V_T was maintained constant. After completion of surgery and before awakening, patients from both groups were allowed to breathe spontaneously while in the semisitting position (head of the bed elevated ~30° from horizontal) before tracheal extubation and PEEP was eliminated in the recruitment group with extubation.

Measurements

Baseline arterial blood gases were obtained at 5 min after anaesthetic induction (in both groups), 5 min after the first recruitment manoeuvre (in the RM group only), at 60 min after tracheal intubation (in both groups), and in the post-anaesthesia care unit at 30 min after tracheal extubation (in both groups). In all patients, blood samples for IL-6

and IL-8 measurements were drawn 5 min after induction and 30 min after tracheal extubation. A non-invasive $NICO_2^{TM}$ monitor (Novametrix Medical Systems Inc., Wallingford, CT, USA) was used to measure the following parameters: (i) end-tidal CO_2, (ii) mean and peak airway pressures, (iii) alveolar minute ventilation [MV_{alv}, calculated as alveolar tidal volume multiplied by ventilatory frequency (alveolar tidal volume=difference between expiratory tidal volume and total airway dead space)], (iv) expiratory tidal volume (V_{TE}), (v) respiratory system dynamic compliance (C_{dyn}, ml cm H_2O^{-1}, calculated as the ratio of the maximum inspiratory volume over the difference between peak inspiratory pressure and PEEP), (vi) airway resistance (R_{aw}, cm H_2O litre^{-1} s^{-1}, computed by least square fittings of the raw waveform data of flow, volume, and pressure to a simple model), and (vii) non-invasive cardiac index by means of a differential form of the Fick equation.

IL-6 was measured by a sandwich enzyme immunoassay (R&D Systems, Minneapolis, MN, USA). In this assay, an anti-IL-6 monoclonal antibody is immobilized onto a microtitre plate. Calibrators, controls, and patient samples are added to the wells. Any IL-6 that binds to the immobilized antibody is detected by a second antibody labelled with alkaline phosphatase. Detection was initiated by adding a colorimetric substrate, followed by absorbance measurements. The absorbance of the calibrators was used to construct a standard curve from which the control and patient results were calculated. The lower limit of detection for this assay is 0.3 pg ml^{-1}. IL-8 was measured by a chemiluminescent immunometric assay on the Immulite automated immunoassay system (Diagnostic Products Corporation, Los Angeles, CA, USA) according to the manufacturer's instructions. The lower limit of detection for this assay is 5.0 pg ml^{-1}.

The intraoperative use of crystalloids and colloids, blood products, and vasopressors was recorded. Medical records were reviewed for postoperative pulmonary complications including deaths. Postoperative respiratory failure was defined as unanticipated mechanical ventilation for >48 h after operation or the need for reinstitution of mechanical or non-invasive ventilation after extubation. Other postoperative pulmonary complications were: (i) acute lung injury [hypoxaemia with a ratio of $Pa_{O_2}/F_{I_{O_2}}$ ≤40 kPa (300 mm Hg) on two consecutive days, new bilateral pulmonary infiltrates, or both, and without evidence of left atrial hypertension], (ii) non-cardiogenic pulmonary oedema, (iii) pneumonia, (iv) atelectasis diagnosed by chest radiogram and requiring bronchoscopic intervention, (v) pneumothorax requiring chest tube placement, (vi) pulmonary embolism, (vii) pleural effusion, and (viii) severe hypercapnia in the recovery room. We also noted the duration of postoperative oxygen use, patients' mental status during the first 24 postoperative hours (normal *vs* confused/delirious) from routine nursing assessments, and duration of hospitalization.

Statistical analysis

The sample size for this study was determined for the primary endpoint of intraoperative $Pa_{O_2}/F_{I_{O_2}}$ ratio. Preliminary data for this endpoint were available from a study that tested the effects of this ventilatory strategy on arterial oxygenation in morbidly obese patients undergoing bariatric surgery.[5] On the basis of the data from this previous study, it was determined that for the endpoint of $Pa_{O_2}/F_{I_{O_2}}$ ratio, a sample size of n=20 patients *per* group would provide statistical power (two-tailed, α=0.05) of greater than 80% to detect a difference between the treatment groups of 10 kPa.

The ratio of Pa_{O_2} to $F_{I_{O_2}}$, ventilatory and haemodynamic parameters, and preoperative and intraoperative variables were summarized and are presented as mean (SD) and median (inter-quartile range) as appropriate. Baseline parameters measured with the $NICO_2^{TM}$ monitor were compared between the experimental groups using t-tests. Data collected after the first RM were compared between the groups using repeated-measures analysis of variance (repeated ANOVA). To supplement the overall repeated measures analysis, groups were also compared at each time point using the two-sample t-test. IL-6 and IL-8 levels, before and after surgery, were compared between the two groups using the rank-sum test as they were not normally distributed. Baseline patient and procedural characteristics and other outcomes were compared across the groups using Fisher's exact test for categorical variables, and the two-sample t-test, or rank-sum test, for continuous variables. In all cases, two-tailed P-values of <0.05 were considered to be statistically significant. Analyses were performed using SAS statistical software (Version 9.1, SAS Institute, Inc., Cary, NC, USA).

Results

Twenty patients were included in each group and all completed the study. Patient characteristics and preoperative comorbidities were similar between the groups, with the exception that more patients in the RM group had documented coronary artery disease (P=0.044) (Table 1). Intraoperative characteristics such as duration of anaesthesia, blood loss, administration of fluids and blood products, need for haemodynamic support with pressors, and intraoperative use of insulin were similar between the two study groups (Table 2, all P>0.10). The number of postoperative days of supplemental oxygen was not different between the groups (P=0.38), nor was the length of hospital stay (P=0.07), although the latter tended to be longer in the recruitment group (Table 2). Five patients in the RM and eight in the control groups developed pulmonary complications (P=0.50) with atelectasis noted on chest radiogram and requiring intervention being the most common complication (Table 3).

Table 1 Patient characteristics and preoperative comorbidities presented as mean (range), mean (SD) or number. *Cystectomy (n=7), prostatectomy (n=7), radical cystoprostatectomy (n=1); †bowel resections (n=5), Whipple (n=9), liver wedge resection (n=3), gastrectomy (n=1), exploratory laparotomy (n=3), cholecystectomy (n=2), large ventral hernia repair (n=2)

Characteristic	Control (n=20)	Recruitment (n=20)
Age (yr)	72.1 (65–88)	73.8 (65–88)
BMI (kg m^{-2}) [mean (SD)]	27.9 (4.4)	27.8 (4.3)
ASA grade (n)		
≤II	6	9
≥III	14	11
Gender (n)		
Female	4	5
Male	16	15
Smoking status (n)		
Current	2	0
Former	9	4
Never	9	16
Preoperative comorbidity (n)		
Cancer	19	16
Coronary artery disease	1	7
Hypertension	9	12
Diabetes mellitus	3	1
Type of surgery (n)		
Lower abdominal operations*	7	8
Upper abdominal operations†	13	12

Table 2 Intra- and postoperative characteristics presented as mean (SD), median (IQR), or number of patients. *Number of patients who received any intraoperative phenylephrine or ephedrine boluses in response to mean arterial pressure ≤60 mm Hg. †Phenylephrine dose represents the mean (SD) from patients who received phenylephrine (control group N=14, recruitment group N=12). ‡Ephedrine dose represents the mean (SD) from patients who received ephedrine (control group N=15, recruitment group N=11). §Recorded as a part of routine nursing assessment for the first 24 postoperative hours. ¶Median (25%–75%)

	Control (n=20)	Recruitment (n=20)
Intraoperative		
Duration of anaesthesia (min)	344 (103)	308 (112)
Fluids administered		
Crystalloid (ml)	3580 (2751–4668)	3351 (2365–4888)
Colloid (ml)	1000 (500–1000)	1000 (500–1000)
Blood (units)	0 (0–2)	0 (0–0)
Blood loss (ml)	550 (237–800)	450 (200–800)
Need for vasopressors (n)*	18	14
Phenylephrine total dose (μg)†	910 (170)	783 (184)
Ephedrine total dose (mg)‡	23 (4)	33 (5)
Phenylephrine infusion (n)	3	3
Postoperative		
Mental status (n)§		
Normal	18	16
Confused/delirious	2	4
Supplemental oxygen (days)¶	3 (1–9)	2 (1–4)
Length of hospital stay (days)¶	8 (7–20)	5 (3–14)

No patient experienced significant haemodynamic compromise [as defined by either the occurrence of hypotension (see Methods) or the administration of a fluid bolus or vasopressor medication to support haemodynamic variables] during a recruitment manoeuvre. There were no differences in intraoperative cardiac indices (Table 4) or of the need for pressors for haemodynamic support (Table 2) between the two groups throughout the course of anaesthesia.

Table 3 Number of postoperative complications. *Some patients had more than one pulmonary complication

Complication	Control	Recruitment
Any pulmonary complication (n)	8	5
Type of pulmonary complication*		
Atelectasis	5	4
Pulmonary oedema	0	1
Pneumonia	1	1
Pleural effusions	4	1
Hypercapnia in the recovery room	1	0
Acute lung injury	1	0
Pulmonary embolism	1	0
Prolonged respiratory failure	1	0
Death (n)	1	1

The recruitment manoeuvre improved intraoperative Pa_{O_2} [mean (SD) acute increase in $Pa_{O_2}/F_{I_{O_2}}$ from 41.5 (12.8) to 54.9 (6.4) kPa (P<0.01)] which remained elevated [54.5 (10.7) kPa, P<0.01] at 60 min after the first RM (Table 5). Conversely, the $Pa_{O_2}/F_{I_{O_2}}$ at 60 min of anaesthesia declined by 15% over the first 60 min [from 47.2 (13.1) *vs* 40 (12.7) kPa in the control group (P=0.018)]. In the recovery room, the $Pa_{O_2}/F_{I_{O_2}}$ was not different between the two groups, and values were not different from their respective baselines (Table 5). As expected, mild hypercapnia was present in the RM group throughout anaesthesia (Tables 4 and 5).

After the recruitment manoeuvre dynamic compliance increased on average 36% and airway resistance decreased on average 21% (Fig. 1). Because of higher PEEP used, the mean airway pressure and peak-inspiratory pressures were significantly higher in the RM group compared with those in the control group (Table 4). Both IL-6 and IL-8 were significantly increased after surgery (Table 6), but the magnitude of increase did not differ between the two groups.

Discussion

The main finding of this study was that compared with a conventional approach to intraoperative ventilation, a ventilatory strategy designed to maintain lung expansion improved intraoperative Pa_{O_2} in the elderly without compromising haemodynamic stability. These effects on Pa_{O_2}, achieved with 40–50% lower minute ventilation, were limited to the intraoperative period. Increases in the serum levels of IL-6 and IL-8 associated with surgery did not depend on intraoperative ventilatory strategy.

Ventilatory strategies designed to maintain lung expansion and minimize the mechanical shear stresses on lung parenchyma have gained popularity in the management of patients with acute lung injury.[6] These involve the use of recruitment manoeuvres to promote reexpansion of atelectasis, followed by ventilation with relatively high PEEP (to prevent reformation of atelectasis) and lower tidal volumes (to minimize mechanical stresses).[11]

Table 4 Mean (SD) intraoperative respiratory variables. Data were analysed using repeated ANOVA. The *P*-value corresponds to the test of the main effect of group (i.e. control *vs* recruitment). To supplement the repeated measures analysis, groups were also compared separately at each time period using the two-sample *t*-test with (*) used to denote significant differences from the control ($P<0.05$). †Increased PEEP (on average 2.5 cm H_2O) in control patients reflects intrinsic PEEP related to the properties of mechanical ventilator. EOS, end of surgery and before the start of spontaneous ventilation; E'_{CO_2}, end-tidal CO_2; V_T, tidal volume; MV, minute ventilation; PIP, peak inspiratory pressure; PEEP, positive end-expiratory pressure

	Baseline	Time (min)					*P*-value
		30	60	90	120	EOS	
Cardiac index (litre m^{-2})							
Control	3.0 (1.1)	3.4 (0.6)	2.9 (1.1)	3.0 (1.2)	3.3 (1.5)	—	0.755
Recruitment	2.5 (0.8)	3.3 (0.7)	3.3 (0.6)	3.2 (0.7)	3.5 (0.8)	—	
Mean P_{aw} (cm H_2O)							
Control	6.5 (0.9)	6.9 (1.1)	6.9 (1.1)	6.6 (0.9)	6.8 (1.0)	7.6 (1.4)	<0.001
Recruitment	6.7 (0.8)	14.4 (0.6)*	14.4 (0.5)*	14.5 (0.7)*	14.5 (0.6)*	14.2 (0.8)*	
PIP (cm H_2O)							
Control	16.8 (3.3)	19.0 (3.3)	19.6 (3.3)	19.5 (3.7)	20.0 (3.9)	21.6 (4.1)	0.009
Recruitment	14.6 (2.5)*	20.9 (1.8)*	21.2 (2.0)	22.1 (2.8)*	21.4 (2.4)	21.3 (2.6)	
PEEP (cm H_2O)							
Control†	2.7 (0.7)	2.6 (0.7)	2.6 (0.8)	2.6 (0.9)	2.4 (0.4)	2.4 (0.5)	<0.001
Recruitment	4.0 (0.4)*	11.8 (0.2)*	12.0 (0.4)*	11.8 (0.2)*	11.9 (0.1)*	11.8 (0.2)*	
MV alveolar (litre min^{-1})							
Control	4.4 (0.9)	4.3 (0.8)	4.1 (0.9)	4.0 (1.0)	4.1 (1.0)	4.9 (1.4)	<0.001
Recruitment	2.3 (0.7)*	2.5 (0.8)*	2.4 (0.7)*	2.7 (0.9)*	3.2 (0.6)*	3.2 (0.8)*	
V_T (exhaled) (ml)							
Control	746 (124)	779 (90)	776 (96)	772 (103)	791 (100)	821 (108)	<0.001
Recruitment	456 (73)*	515 (105)*	489 (66)*	495 (80)*	521 (64)*	529 (74)*	
E'_{CO_2} (kPa)							
Control	5.0 (0.5)	4.8 (0.5)	4.9 (0.5)	5.0 (0.6)	5.0 (0.7)	5.1 (0.7)	<0.001
Recruitment	5.6 (0.6)*	6.1 (0.7)*	6.2 (0.6)*	6.4 (0.8)*	6.2 (0.8)*	6.4 (0.7)*	

Table 5 Mean (SD) intra- and postoperative oxygenations and ventilation. *Two sample *t*-test $P<0.05$ for recruitment *vs* control. †Paired *t*-test $P<0.05$ for within-group comparison *vs* baseline. ‡Elevated Pa_{CO_2} in the recruitment group at baseline can be attributed to lower tidal volume used in this group

	Control ($n=20$)	Recruitment ($n=20$)
Pa_{O_2}/FI_{O_2} (kPa)		
Baseline	47.2 (13.1)	41.5 (12.8)
60 min	40.0 (12.7)†	54.5 (10.7)†,*
Recovery room	44.8 (15.7)	40.0 (7.6)
Pa_{CO_2} (kPa)		
Baseline‡	5.3 (0.6)	6.2 (0.6)*
60 min	5.4 (0.6)	6.8 (0.7)†,*
Recovery room	6.0 (0.9)†	6.4 (0.6)

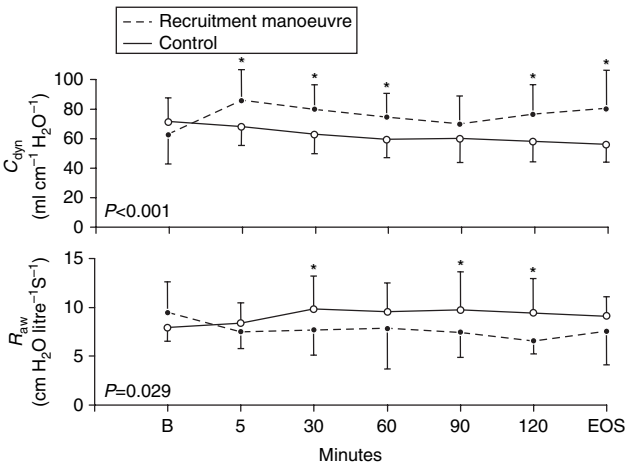

Fig 1 Dynamic respiratory system compliance (C_{dyn}) and airway resistance (R_{aw}) in the control and recruitment groups. C_{dyn} increased and R_{aw} decreased after recruitment and remained greater compared with control patients throughout the operation. Data are mean (SD). *$P<0.05$ the control *vs* the recruitment group at the same time intervals. *P*-value is overall statistical significance from ANOVA. B, baseline measurement before recruitment manoeuvres. EOS, end of surgery and before initiation of spontaneous ventilation.

Prevention of atelectasis also minimizes mechanical stresses caused by the repetitive opening and closing of lung units.[7] The goal of these strategies is to maximize the gas exchange and to minimize the risk of ventilator-induced lung injury. Both animal models[12 13] and human studies[6] of acute lung injury suggest that these strategies can accomplish both goals in some settings, although the early promise of dramatic improvement in the survival of these patients has not been realized outside the intensive care unit.[6]

The principles elucidated in these studies of lung injury have been applied to patients with normal lungs who require anaesthesia and surgery. Like patients with acute lung injury, normal patients develop areas of atelectasis shortly after the induction of anaesthesia,[1 2] and recruitment strategies can partially reverse these changes.[3] We have shown that a strategy similar to that used in the current study significantly increased Pa_{O_2} and respiratory system compliance in morbidly obese patients.[4 5] Although, as in the present study, we did not directly image atelectasis, changes in these surrogates are both consistent with reductions in the quantity of atelectasis. The changes in dynamic compliance and Pa_{O_2} in the present study were well coupled (Fig. 1, Table 5), that is,

Table 6 Median (IQR) cytokine concentrations at baseline and in recovery room. All comparisons for control *vs*. recruitment at baseline and in the recovery room were $P>0.05$ (rank-sum test); $^{\dagger}P<0.01$(signed rank test) for within-group comparing change from baseline (recovery room minus baseline) to zero for each group

	Control ($n=20$)	Recruitment ($n=20$)
IL-6 (pg ml^{-1})		
Baseline	3.3 (1.9–9.7)	2.8 (1.6–5.4)
Recovery room	113.0 (50.2–214.5)	70.0 (9.2–177.5)
Delta	109.6 (47.0–215.0)†	69.2 (7.4–175.0)†
IL-8 (pg ml^{-1})		
Baseline	7.1 (5.1–12.3)	7.0 (5.0–12.0)
Recovery Room	18.4 (6.3–60.7)	29.7 (5.0–83.2)
Delta	11.1 (1.0–49.0)†	22.5 (0.0–73.1)†

higher compliance coincided with higher Pa_{O_2}/FI_{O_2} ratios at 60 min after recruitment. We have previously demonstrated that the use of high PEEP intraoperatively does not guarantee maintenance of lung reexpansion.[4 5] A gradual decrease in compliance during surgery suggests reoccurrence of lung collapse and indicates when the recruitment should be repeated. The timing of rerecruitment intervals in our patients was based on our previous study where we more frequently monitored changes in compliance and Pa_{O_2}.[4] Also consistent with this previous work,[4] improvements in Pa_{O_2} quickly dissipated after the conclusion of anaesthesia, such that Pa_{O_2} in the recovery area was similar in the two experimental groups. In contrast, several studies demonstrated that improved Pa_{O_2} and lung mechanics achieved by lung recruitment may extend beyond the immediate intraoperative period.[14 15]

Although the benefits of higher partial pressures of oxygen are clear, it is less clear whether conventional ventilation strategies produce subclinical lung injury that could potentially be avoided with minimized atelectasis and lower tidal volumes. Choi and colleagues[16] found that in patients with normal lungs undergoing prolonged abdominal operations, the use of smaller tidal volumes (6 ml kg^{-1}) with PEEP prevented increases in procoagulant factors in bronchoalveolar lavage fluid indicative of lung injury that were associated with larger tidal volumes (12 ml kg^{-1}) in the absence of PEEP. However, in a similar comparison of ventilatory strategies applied for 6 h in the intensive care unit post-cardiopulmonary bypass, Wrigge and colleagues[17] found no differences in serum markers of inflammation, IL-6 and IL-8, and only small changes in bronchoalveolar lavage concentrations of TNF-α. Of note, IL-6 and IL-8 are non-specific markers of both surgical and ventilator-induced lung injuries. In another study, ventilation for 1 h with very high tidal volumes (15 ml kg^{-1} ideal body weight and ZEEP) in patients without pre-existing lung injury did not increase systemic levels of inflammatory mediators.[18] Similarly, we found that increases in IL-6 and IL-8 associated with surgery did not depend on ventilation mode. This result is consistent with other reports that recruitment manoeuvres, which apply transient high mechanical stresses to lung parenchyma, do not produce evidence of systemic inflammation.[19] Thus, we find no evidence that these two ventilation modes differentially affected inflammatory responses.

Maintenance of lung expansion requires higher mean airway pressures, and we were concerned that the haemodynamic consequences would not be well tolerated in these elderly patients, especially as decreases in chest wall compliance with age[20] may necessitate higher intrathoracic pressures to accomplish expansion of atelectasis. However, there were no differences in haemodynamics between the groups, and no episode of acute haemodynamic compromise during recruitment manoeuvres, suggesting that this strategy is well tolerated.

In the control group, oxygenation was adequate to prevent desaturation in these patients without the use of recruitment manoeuvres, high PEEP, and low tidal volumes, so does an 'open lung' ventilatory strategy in this patient population provide a clinical advantage? There is increasing evidence that a high intraoperative Pa_{O_2} is beneficial. For example, administration of perioperative supplemental oxygen during colorectal operations reduces two-fold the rate of wound infections,[21] an effect presumably mediated through an increase in tissue oxygen tension which may facilitate oxidative killing by the production of superoxide radicals.[22] Similarly, supplemental intraoperative oxygen administration reduces the incidence of postoperative nausea or vomiting by approximately half, perhaps through reduction of subtle intestinal ischaemia.[23] Furthermore, our ventilation strategy was associated with mild permissive hypercapnia, which may improve intestinal perfusion and oxygen tension in various tissues.[24] Hypercarbia may also be associated with better postoperative cognitive scores compared with patients subjected to normo- or hypocarbia.[25] Our two groups did not differ in the number of patients who were assessed by nursing staff as confused or delirious, but we did not perform any detailed neurocognitive testing.

This study has several limitations. We examined several outcome variables such as duration of hospitalization, and number of postoperative pulmonary complications that were favourable to the recruitment group, but the number of patients studied is not sufficient to draw conclusions. Secondly, systemic cytokine levels are at best a crude measure of lung inflammation, especially in the face of concurrent changes caused by surgical injury. For example, Wolthuis and colleagues[26] demonstrated attenuated pulmonary levels of IL-8 with protective ventilation, although there was no difference in plasmatic levels of IL-6 and IL-8 between protective and non-protective ventilatory strategy. Therefore, we cannot conclusively comment on the possibility for existence of subclinical lung injury in either of our groups. Finally, although we found no evidence of adverse events associated with the recruitment strategy, we did not have sufficient numbers of patients to comment on the potential risk low-frequency events such as pneumothorax.

In conclusion, a lung recruitment strategy in elderly patients results in improvement of intraoperative Pa_{O_2} and lung mechanics with lower minute ventilation during laparotomy. This exploratory study is only designed to test the feasibility of this mode of ventilation, and further studies are necessary to explore whether this ventilatory strategy improves clinically relevant outcomes in elderly patients.

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